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studies to liver membranes were carried out with the purpose of investigating the mechanism of action of also observed when rabbits previously made hypercholesterolaemic by being fed casein for 4 weeks were mg/dl-1 to 77 +/- 6 mg/dl-1. By contrast the animals receiving FCE 27677 (10 mg kg-1 d-1) mixed with New Zealand White rabbits were fed with casein for 4 weeks, LDL cholesterol increased from 14 +/- 3 systemic acylCoA:cholesterol acetyltransferase (ACAT, EC 2.3.1.26) inhibitor, has been evaluated. When phenyl]-N'-[(4R,5R)-2-(4-dimethylaminoph eny l)-4,5-dimethyl-dioxolan-2-yl]methylurea) a novel potent elevation of low density lipoprotein (LDL) that is caused by oversecretion of apoB-100 containing Rabbits fed a wheat starch casein diet develop hypercholesterolaemia characterized by the plasma FCE 27677, was respectively 10.5, 22.4, and 12.5 mg kg-1 d-1. The turnover rate of [125I]LDL in the the drug. The LDL apoB-100 production rate in chow-fed, casein-fed, and casein-fed rabbits receiving then treated for a month with FCE 27677. [125I]LDL plasma turnover studies and [125I]LDL binding the casein diet maintained a normal LDL concentration (22 +/- 3 mg dl-1). This hypolipidaemic effect was lipoprotein (VLDL)-LDL receptor. In this study, the effect of FCE 27677 ((-)N-[2,6-bis(1-methylethyl) lipoproteins by the liver and by the suppression of the EDTA-sensitive hepatic beta-very low density

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ACAT inhibitor

of the apoB-100 synthetic rate. suppressed by casein feeding. Altogether the results are consistent with the idea that FCE 27677 by acting inhibitors may be useful drugs for the treatment of human dyslipoproteinaemia secondary to derangement through inhibition of the cholesterol esterification in the liver normalizes the LDL synthetic rate. ACAT 27677 did not normalize the activity of the hepatic beta-VLDL-LDL EDTA-sensitive receptor which is (2.381 vs 2.079 pools d-1). Both values were lower than that in chow-fed animals (3.271 pools d-1). FCE animals receiving the drug was not, however, different from that in the rabbits fed the casein diet alone

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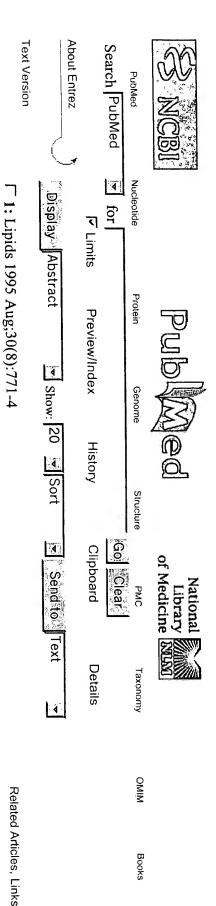
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cells: evaluation of E5324, an acyl-CoA cholesterol acyltransferase inhibitor Inhibition of cholesterol esterification in macrophages and vascular smooth muscle foam

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arterial smooth muscle cells. cholesterol esterification and CE mass in atherosclerotic foam cells, derived from either macrophages or significant measure of the efficacy of drugs designed to modulate cholesterol metabolism. E5324 aortic smooth muscle cells and in macrophages. In addition, E5324 reduced the cellular mass of CE, the macrophages and in smooth muscle cells. E5324 significantly inhibited cholesterol esterification in rat absorbable ACAT inhibitor, to inhibit esterification of fatty acids to cholesterol and CE accumulation in (n-butyl-N'-[-2-[3-(5-ethyl-4-phenyl-1H-imidazol-1-yl)propoxy]-6- methyl-phenyl]urea), a novel, orally major enzyme responsible for esterification of intracellular cholesterol. We evaluated the ability of E5324 smooth muscle cells of the atherosclerotic lesion. Acyl-CoA cholesterol acyl-transferase (ACAT) is the Cholesteryl esters (CE) comprise the principal lipid class that accumulates within macrophages and for efflux in vivo (reverse cholesterol transport). These results demonstrate that E5324 can inhibit treatment of lipid-loaded smooth muscle cells reduced CE mass by 29%. Although free cholesterol treatment of macrophages exposed to acetylated low-density lipoprotein reduced CE mass by 97%, and increased approximately twofold, this free cholesterol would presumably be accessible to the membrane

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